11:45

785-6 Diastolic Function is Impaired in Uncomplicated Obesity

Gian F. Mureddu, Giovanni de Simone, Rosanna Greco, Giuseppe F. Rosato, Franco Contaldo *Federico II University, Napoli, Italy*

As a part of a project designed to assess mechanisms underlying heart failure in obesity, 38 normotensive, uncomplicated, white obese subjects [14 men, 24 women; age 35 ± 13 years; body mass index (BMI) = 36 ± 6; blood pressure (BP) = 124.5 ± 14.2/79.3 ± 9.3 mmHg] have been studied by Doppler Echocardiography and compared with a group of 39 normotensive, normal-weight, white volunteers (16 men, 23 women, mean age 36 ± 12 years; BMI = 22 ± 3; BP-119.8 ± 14.5/71.8 ± 9.8). Left ventricular (LV) mass (M) normalized by height$^{2.7}$ (LVMI), fractional shortening (FS) and several measurements of diastolic function were compared by ANOVA after adjusting (ANCOVA) for potential confounders (sex, age, systolic and diastolic BP) identified by a correlation matrix. FS was comparable between groups (obese = 29 ± 5%; normal-weight = 30 ± 7%). Obese patients exhibited higher LVMI than normal-weight subjects (38 ± 11 vs 26 ± 8 g/m$^{2.7}$; $p < 0.0001$), lower transmitral peak E wave velocity (E) (64.4 vs 73.6 cm/sec; $p < 0.0001$), prolonged isovolumic relaxation time (IVRT) (81.6 vs 59.6 msec: $p < 0.0001$) and deceleration time of E wave (DTE) (172.4 vs 147.2 msec: $p < 0.0002$), normal transmitral peak A velocity (A). E/A flow velocity ratio was lower in obese than in normal-weight subjects (1.16 vs 1.40; $p < 0.03$) and atrial filling fraction (AFF) was higher (34 ± 1% vs 30 ± 1%; $p < 0.05$). While with univariate analysis LVMI was related directly to DTE ($r = 0.50$; p 0.0001), and IVRT ($r = 0.35$; $p < 0.004$) and inversely to E ($r = -0.45$; $p < 0.0001$) and E/A ratio ($r = -0.36$; $p < 0.001$) with ANCOVA between group differences in IVRT and E velocity were independent of LVMI. Differences in DTE, E/A and AFF disappeared when controlling for LVMI. Thus, even uncomplicated obesity is associated with impairment of the early active relaxation phase, independently of levels of LVMI, paralleling abnormalities of early filling flow. Prolonged DTE and increased AFF might be compensatory mechanisms associated with increased LVM.

786 Evaluation of Coronary Flow in Humans

Wednesday, March 22, 1995, 10:30 a.m.–Noon
Ernest N. Morial Convention Center, Room 61

10:30

786-1 Dynamic Detection of Coronary Stenosis by Doppler-tipped Guidewire

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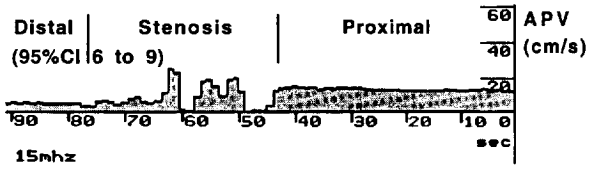
The accuracy and reliability of intracoronary Doppler measurements were thought to depend in part on the site of measurement and to the position of the sensor relative to the arterial wall and size. To obviate this drawback Doppler velocity measurements were performed during a constant mechanically-driven pull-back at a speed of 1 mm/sec from the distal to the proximal segment of coronary arteries. Coronary blood flow velocity was measured using a 15 MHz Doppler-tipped 0.014-in guidewire with a plot trend recorded throughout the pull-back. Continuous assessment of the time-average peak velocity (APV, cm/sec) was performed in 10 male single-vessel diseased patients, aged 56 ± 9 years, submitted to PTCA for a 79 ± 15 percent stenosis diameter of the left anterior descending (n = 6) or the left circumflex coronary (n = 4) artery. The dynamic velocity measurements were also performed in the other angiographically normal left coronary artery for comparison with the trend in the stenotic arteries. APV was measured each second in the normal segments to calculate 95% confidence interval (CI). If the APV was ≥3 times outside the limits of CI when crossing the stenosis, the lesion was considered significant. Coronary angiogram was used as a reference for the assessment of sensitivity and specificity. *Results:* In the angiographically normal arteries and in the non-diseased segments APV varied smoothly. On the contrary, in the stenosed segments APV varied sharply depicting a "Manhattan-like" tracing (figure).



Conclusions: 1. During pull-back of Doppler-tipped guidewire, velocity remains stable in non stenotic coronary segments. 2. Stenosis is associated with high changes in velocity which may reflect turbulent flow. 3. Dynamic velocity assessment may be useful for the detection of coronary stenosis.

10:45

786-2 Coronary Flow Reserve vs Translesional Velocity Gradient by Doppler Guidewire in Assessing Intermediate Coronary Stenoses

James D. Joye, David Lasorda, Tony Farah, Bryan C. Donohue, Douglas S. Schulman *Medical College of PA, Pittsburgh, PA*

Intracoronary Doppler guidewire measures of coronary flow reserve (CFR) and translesional velocity gradients (TVG) have been used to determine the physiologic significance of intermediate coronary lesions (40–70%). In many patients with intermediate stenoses CFR and TVG values are discordant, raising the possibility that CFR is diminished due to microvascular rather than epicardial disease. In 13 patients with an intermediate coronary lesion, we used an intracoronary Doppler guidewire to record baseline coronary flow velocity proximal and distal to each lesion, thus defining TVG. Peak hyperemic velocity was then recorded distally after an intracoronary bolus of adenosine to obtain CFR. All patients had decreased CFR (mean = 1.4 ± 0.3; normal ≥2.0) but trivial TVG (mean = 1.3 ± 0.3; normal <1.7). Each patient had a reversible perfusion defect on stress SPECT ^{201}Tl imaging and subsequently underwent PTCA. These patients were asked to return at 6 months for a repeat stress SPECT ^{201}Tl test. Two patients had restenosis prior to repeat stress testing. The remaining 11 patients had their baseline and 6 month post-PTCA scans analyzed.

Region of Interest ^{201}Tl Quantitation (counts as % of normal)

Pre-PTCA	Stress	77 ± 2.5	Post-PTCA	Stress	89 ± 3.0*
	Delay	93 ± 3.0*		Delay	93 ± 4.0

P(ANOVA) < 0.0001 (*p < 0.05 vs Pre-PTCA Stress)

All 11 patients demonstrated qualitative normalization of SPECT ^{201}Tl scans at 6 month follow-up. Quantitative polar mapping and region of interest analysis confirmed the improvement in perfusion. Therefore, blunted CFR was related to a physiologically significant intermediate stenosis rather than a microvascular cause. It appears that CFR is a more reliable indicator of lesion significance than TVG.

11:00

786-3 Adenosine Causes Flow-Mediated Epicardial Vessel Dilation in Humans

Geoffrey A. Rose, Michael A. Mathier, Sudhir S. Kushwaha, Marc J. Semigran, Robert E. Dinsmore, Michael A. Fifer *Massachusetts General Hospital and Harvard Medical School, Boston, MA*

The predominant effect of intracoronary adenosine (Ad) is vasodilation of the microcirculation, but Ad also increases epicardial coronary artery diameter. It is not known whether epicardial dilation is a direct action of Ad or an indirect effect mediated by increased flow or shear stress. We reasoned that changes in the diameter of the left anterior descending (LAD) artery *upstream* to the site of Ad delivery must reflect an indirect effect, whereas *downstream* vasodilation would reflect the sum of direct and indirect effects. We therefore compared changes in upstream and downstream LAD diameter in 11 patients without significant coronary artery disease in whom LAD flow increased at least three-fold (n = 6) or remained unchanged (n = 5) in response to Ad 10^{-4} M infused via a 3F Doppler catheter. LAD diameter was assessed by quantitative angiography 2.5 mm upstream and 2.5 mm downstream to the site of Ad delivery. LAD flow was calculated as the product of downstream cross-sectional area and Doppler velocity. LAD diameter (mm) is shown as mean ± standard error of the mean.

	Flow Increased			Flow Unchanged		
	Baseline	Adenosine	p	Baseline	Adenosine	p
Downstream	2.08 ± 0.22	2.48 ± 0.30	0.06	1.97 ± 0.16	1.99 ± 0.14	0.16
Upstream	2.38 ± 0.20	2.65 ± 0.21	0.004	2.28 ± 0.26	2.18 ± 0.26	0.23

Thus, epicardial vessel dilation in response to Ad occurs only in the presence of an increase in coronary blood flow, and there is significant dilation upstream from the site of Ad infusion.

Conclusions: Adenosine causes flow-mediated epicardial vessel dilation in humans, with little or no direct effect on epicardial vessel diameter.